

## Environment, disease and mortality in early Virginia

Carville Earle

---

Early Virginia (1607-24) was a nightmarish world of disease and death, perhaps unsurpassed in the annals of English colonization. Typhoid fever and dysentery visited Jamestown in recurrent epidemics killing 30 per cent or more of the colonists with each onslaught. Yet Jamestown endured because the leaders of the Virginia Company misapprehended the nexus between the estuarine environment and water-borne, non-immunizing diseases. Each summer, death stalked the town as invading salt water pushed up the estuary and concentrated pathogens in the town's water supply. The prevention of disease and death required the abandonment of Jamestown and relocation into healthier niches, which occurred with the dissolution of the Virginia Company in 1624.

The English colonization of North America resembled an epic geographical comedy. Captivated by the facile belief that latitude controlled climate and environment, the English mind transfigured America into a pristine, temperate Europe.<sup>[1]</sup> Along the fresh face of the Atlantic Seaboard of North America, they envisaged familiar Old World environments—environments of moderation, of temperate maritime climate consisting of more humid lands to the north in what they would call, after their analogue, New England, and Mediterranean-like lands in the latitudes of Virginia and the Carolinas. Here was a premise for uproarious comedy and, at times, tragedy.

Seldom has such a misguided theory produced so many bizarre consequences. In the latitude of Spain, the English colonists dutifully tended the vineyards that yielded insipid wine. In the latitude of France, the settlers tried valiantly to produce silk, but the imported worms, reared on the white mulberry of Europe, refused to feed on the abundant reds of America. And Virginians watched in amusement as Polish potash-makers, especially imported from the dense stands of pine in the European plain, wandered aimlessly in search of the infrequent and scattered pines in early Virginia.<sup>[2]</sup> Countless tales of this sort of blundering comically relieve the

[1] Gary Dunbar, Some curious analogies in explorers' preconceptions of Virginia *Virginia Journal of Science*, New Series 3 (1958) 323-6; H. Roy Merrens, The physical environment of early America: images and image makers in colonial South Carolina *Geographical Review* LIX (1969) 530-56; Charles M. Andrews *The colonial period of American history* (4 vols; New Haven: Yale University Press 1964); and Carville V. Earle The first English towns of North America *Geographical Review* LXVII (1977) 34-50

[2] In addition to the sources cited above, see Irene W. D. Hecht, The Virginia colony, 1607-1640: a study in frontier growth (unpubl. Ph.D. dissertation, Univ. of Washington 1969); and Edmund S. Morgan The labor problem at Jamestown, 1607-18 *American Historical Review* LXXVI (1971) 595-611

story of relentless English expansion during the seventeenth century, yet on occasion the tales cross the fine line separating comedy from tragedy. In the nightmarish world of early Virginia, these fanciful environmental theories conspired with short-sighted geographical policies to produce levels of disease and mortality unsurpassed in the annals of English colonization.

English colonization was writ large in Virginia, the first permanent English settlement in North America. Wrong-headed environmental models plagued the colony from the very beginning in 1607. Colony officials in England drew clever analogies between the European and American environments while glossing over decisive differences in detail. To the misfortune of Virginia and her settlers, the American mainland was a lustier and more robust land than Europe.<sup>[1]</sup> Maritime Europeans were unaccustomed to the seasonal extremes of the continental climates of North America. New World winters were colder and snowier, and summers were hotter. River and stream levels also fluctuated radically, swelling after the winter melt and spring rains and receding markedly during the scorching American summers. The English also discovered that American rainstorms were more intense, thunder more violent, lightning more flashing, and all were more frequent. Other environmental details were also dissonant with the Englishmen's maritime model of America. American forests, for instance, contained an enormous diversity of species and lacked the large, homogeneous stands of maritime Europe, while American precipitation, which in theory should have declined toward the tropics, in fact increased substantially. The English were slow learners. Gradually they abandoned their maritime model and their latitudinal analogies for a more realistic assessment of the New World environment, in the process leaving us a bountiful supply of amusing anecdotes. But it was in Virginia that the maritime model stood the first test, and the learning was painful indeed. The Company leadership could not comprehend the enormous environmental changes taking place along the James River estuary between spring and late summer, and hence they obstinately insisted that the colony remain centred at Jamestown which was located in one of the most dynamic and deadly niches of a robust and lustier land.

Disease has long been suspected as one of the principal causes of death in early Virginia, with Jamestown as the locus. This association of place, disease and mortality, although often observed, has seldom been understood. This essay reveals how rather remarkable seasonal changes in the estuarine environment and the Virginia Company's misunderstanding of these changes contributed to death from water-borne enteric diseases. A geographical model of disease mortality accounts for spatial, seasonal and annual mortality variations in Jamestown and the James River estuary between 1607 and 1624. The model, derived from the first year in Jamestown, suggests the probable causes of disease-related deaths and offers a logical and consistent account of the pathogenic organisms; the sources of infection; the incidence of infection, morbidity and mortality; and the recurrence of epidemics. The plan of the essay entails: (1) the derivation of the model based on the first year at Jamestown; (2) its application and testing for the years 1608-24; and (3) a discussion of the Virginia Company's inability to understand the New World environment and thereby to lower mortality rates and some speculations

[1] Carl O. Sauer, The settlement of the humid east *Climate and Man*, *Yearbook of Agriculture*, 1941 (Washington, D.C.: Government Printing Office 1941) 157-66, Donald W. Meinig, The American colonial era: a geographical commentary *Proceedings, Royal Geographical Society of Australia, South Australian Branch* LIX (1958) 1-22

on the causes of declining mortality after 1624 in Virginia and the English colonies generally.

The first year in Virginia portended the dreadful mortality that ravaged Virginia until 1624. Things went well at first. The expedition of three vessels and 144 persons left England in December 1606, headed south and west to the West Indies, and then north to the Chesapeake Bay, entering it on 26 April 1607. Shortly thereafter, the colonists established Jamestown on the north side of the James River, nearly 50 miles from its mouth.<sup>[1]</sup> The Virginia spring was beneficent, and when Captain Newport departed on 22 June he left 104 healthy colonists. But by late summer, the colony took on a more somber attitude. On 6 July, George Percy's journal mentioned the death of one John Asbie by the "bloudie fluxe", and 3 days later George Floure died of the "swelling". In the space of 1 month, 21 colonists died causing Percy to lament that "our men were destroyed with cruell diseases, as swellings, Flixes, Burning Fevers, and by warres, and some departed suddenly, but for the most part they died of meere famine . . .".<sup>[2]</sup> Forty-six were dead by the end of September, and in January when the first supply arrived in Virginia, just 38 of 104 colonists were barely alive.<sup>[3]</sup>

The abundance of death demanded an explanation. But Percy's speculation that "meere famine" was the cause of death is unconvincing. In support of his thesis, the colonist's daily ration consisted of just half a pint of wheat and another of barley, mixed in a gruel, and yielding roughly one-half the caloric intake required for an active man of the colonists' stature.<sup>[4]</sup> But we should not hastily accept Percy's "meere famine", if only because of the political disputes and intrigues rending the first colony. Percy may have had an axe to grind. The selection of Jamestown over the Archer's Hope site had displeased him, and he may have cast his support to the faction accusing President Wingfield of hoarding the colony's food and drink for presidential favourites. Whatever Percy's motives, his emphasis on famine spotlighted attention on President Wingfield. The President, of course, denied these allegations.<sup>[5]</sup> His rebuttal draws indirect support from one of his enemies, Captain John Smith. Smith made little of the shortage of provisions, stating matter-of-factly on several occasions that the colony still had so many weeks of supplies remaining. He knew that the colony had a supplementary source of food in the annual sturgeon run. Thousands of these fish entered the James estuary in April and May, and their run to freshwater spawning grounds continued through the summer, when the big fish came in. "From the end of May till the end of June," wrote Smith, "are taken few but young Sturgeons of 2 foot or a yard long. From thence till the midst of September, them of 2 or three yards long and few others. And in 4 or 5 houres with one nette were ordinarily taken 7 or 8:

[1] Observations of Master George Percy, 1607, pp. 5-23 of Lyon Gardiner Tyler (Ed.), *Narratives of Early Virginia, 1606-1625* (New York: Charles Scribner's Sons 1907); on the selection of Jamestown, see Earle, "The first English towns", 34-50

[2] Observations of Master George Percy 20. One hundred and five colonists are claimed in "The Proceedings of the English Colony", *Travels and Works of Captain John Smith I*, Edward Arber (Ed.), Burt Franklin: Research and Source Work Series no. 130 (2 parts; New York: Burt Franklin, originally published 1910) 94

[3] Arber, *Travels and works I* lxxvi 9, 95. Alexander Brown, *The first republic in America* (New York: Houghton Mifflin Company 1898) 55

[4] Herbert Renardo Cederberg, Jr, An economic analysis of English settlement in North America, 1583-1635 (unpubl. Ph.D. dissertation, University of California 1968) 144. Arber, *Travels and works II* 391-2

[5] A Discourse of Virginia per: Ed: Ma: Wingfield, Arber, *Travels and works I* lxxiv-xci

often more."<sup>[1]</sup> A few years later, Rolfe related that two men in a few hours axed forty sizable sturgeon.<sup>[2]</sup> As the Atlantic sturgeon averages above 100 pounds, the output of two axe-wielding men would have totalled 4,000 pounds, or nearly 40 pounds per colonist per day.<sup>[3]</sup> A daily intake of 2 pounds of sturgeon, some sea crabs, and the wheat-barley gruel was more than adequate for the colonists' metabolic needs. Furthermore, 2 pounds of fish daily provided 90 per cent of the daily thiamine requirement, thus thwarting an outbreak of beriberi, which has been postulated by the medical historian Blanton.<sup>[4]</sup>

The food supply during Jamestown's first summer, though unappealing, provided sufficient nutriture to ward off starvation and vitamin deficiency diseases. Starvation did not cause death but the possibility constantly played on the fears of the colonists. By mid-September, the colonists perceived that starvation was imminent. Newport had left them supplies for 13 or 14 weeks; and even allowing for increased food per capita by the death of 50 colonists, by September they had only enough supplies for 4-8 weeks, the sturgeon run was falling off, and they did not expect additional supplies until October at the earliest.

Although Percy blamed famine for Virginia's troubles, his list of clinical symptoms brings us closer to the actual causes of death—typhoid, dysentery and perhaps salt poisoning. Medical historians are generally agreed that Percy's "flices" or "bloudie Flices" describe dysentery, while "Burning Fevers" are symptomatic of typhoid fever.<sup>[5]</sup> The "Swellings", though they may be associated with dysentery, could also result from salt intoxication from the salty river water.<sup>[6]</sup> These three diseases are also indicated by the incidence and rapidity of death, as chronicled by Percy. Typhoid fever progresses rapidly from infection by the bacterium, *Salmonella typhi*. The first week may be symptomless, as the organisms spread through the bowel wall and into the lymphatic glands. In the second week, the organism enters the bloodstream, causing a rapid rise in body temperature, recognized by colonials as the "burning fever". The illness peaks in the third week, and death may result. Before the use of antibiotics, it is estimated that 15-20 per cent of

[1] A Discourse of Virginia per: Ed: Ma: Wingfield Arber, *Travels and works* I 8-9, 51

[2] Philip A. Bruce, *Economic history of Virginia in the seventeenth century*, I (2 vols; New York: Macmillan and Co. 1896) 112

[3] Edward C. Raney, Freshwater fishes, *The James River Basin, Past, Present, and Future*, Virginia Academy of Science, James River Project Committee (Richmond: 1950) 154 James Wharton, *The bounty of the Chesapeake: fishing in colonial Virginia*, Jamestown 350th Anniversary Historical Booklets, No. 13 (Williamsburg: Virginia 350th Anniversary Celebration 1957)

[4] H. L. A. Tarr, Changes in nutritive value through handling and processing procedures *Fish as Food* II (4 vols; New York: Academic Press 1961-5) 248; Wyndham B. Blanton, Epidemics, real and imaginary, and other factors influencing seventeenth century Virginia's population *Bulletin of the History of Medicine* XXXI (1957) 454-62

[5] Very useful are John Duffy, *Epidemics in colonial America* (Baton Rouge: Louisiana State University Press 1953); Wyndham B. Blanton, *Medicine in Virginia in the seventeenth century* (Richmond: The William Byrd Press Inc. 1930) 3-77; Thomas P. Hughes, *Medicine in Virginia, 1607-1699*, Jamestown 350th Anniversary Historical Booklets (Williamsburg: Virginia 350th Anniversary Celebration Corporation 1957); Richard Harrison Shryock, *Medicine and Society in America, 1660-1860* (New York: New York University Press 1960) 82-116

[6] Hans G. Keitel, *The pathophysiology and treatment of body fluid disturbances* (New York: Appleton-Century-Crofts 1962) 162-4; John H. Bland, Clinical physiology and four avenues of loss and gain *Clinical Metabolism of body water and electrolytes* (Philadelphia: W. B. Saunders Company 1963) 133-64



infected persons died.<sup>[1]</sup> Dysentery, caused by amoebic parasites, produced the "bloody flux". While several types of amoebic parasites reside in the human intestinal tract, most are harmless commensals or they bring on diarrhea or mild dysentery. More dangerous is *Endamoeba histolytica*, which may invade the bowel wall, causing ulceration and the bloody stools which gave the disease its seventeenth-century name. More serious complications arise when these parasites bore into a large blood vessel causing massive hemorrhage or the amoeba get into the bloodstream and travel to other organs. Dysentery is often fatal, especially when populations are weakened by other illnesses or undernourishment. Pre-antibiotic mortality rates of 12–25 per cent have been recorded.<sup>[2]</sup> Like typhoid, dysentery can act quickly, though the rates of incubation vary with the individual. "In controlled experiments with human volunteers Walker and Sellards found that the prepatent period, i.e., from exposure until the amebas appeared in the stools, averaged nine days, varying from one to 44 days in the 17 of 20 exposed individuals who became infected."<sup>[3]</sup> Clinical symptoms usually appear within 1–4 weeks, but the range may be from a few days to several months.

An epidemic of typhoid fever and dysentery is consonant with Percy's description of death and sickness at Jamestown. Percy first noted disease-related deaths on 6 July and sickness and death continued "for the space of sixe weekes"—a time span in keeping with the progression of typhoid fever and dysentery. Furthermore, 50 of the 104 colonists had perished by mid-September. The actual mortality rate of 48 per cent exceeds just slightly the sum of expected pre-antibiotic mortality rates for typhoid (15–20 per cent) and dysentery (12–25 per cent), or a total rate of 27–45 per cent. Our estimated rate of mortality may be criticized as too high on the grounds that some colonists died of both pathogens, and therefore the separate rates are not additive. The evidence does not permit an estimate of multiple causes of death. On the other hand, my guess is that the correct estimate of disease mortality probably lies in the 27–45 per cent range, and near the upper end. This supposition is based on several considerations. First the Jamestown population was probably under 30 years of age, and hence included the most susceptible age groups for typhoid (15–25 years) and dysentery (26–30 years).<sup>[4]</sup> Furthermore, the concentrated and confined population facilitated the spread and incidence of these

[1] The fever lasts 21 days usually, and occasionally up to 33 days. Frederick P. Gay, *Typhoid fever considered as a problem of scientific medicine* (New York: The Macmillan Company 1918) 13–24; Gay shows 15–27 per cent mortality for the London Fever Hospital, 1848–70. See also William Budd, *Typhoid fever: its nature, mode of spreading, and prevention* (New York: n.p. 1931), Jacques M. May, *The ecology of human disease* (New York: MD Publications, Inc. 1958), 171–88. In general, I agree with Jones that typhoid fever killed numerous Virginians in 1607. I disagree with him on the following points: (1) that typhoid, aided by beriberi, was the principal killer; (2) that typhoid was probably introduced by Reverend Robert Hunt; and (3) that the Jamestown environment was essentially passive in the typhoid epidemic. Gordon W. Jones, The first epidemic in English America *Virginia Magazine of History and Biography* LXXI (1963) 3–10.

[2] Ernest Carroll Faust, *Amebiasis*, Publication No. 191, A Monograph in American Lectures in Internal Medicine (Springfield, Illinois: Charles C. Thomas, 1954). Parasitism in South-eastern United States: a symposium *Public Health Reports* 70 No. 10 (1955) 957–75; W. B. Blanton, *Medicine in Virginia in the seventeenth century*, 63; Arthur L. Bloomfield, A bibliography of internal medicine: amebic dysentery *Journal of Chronic Diseases* V (1957) 235–52; May, *The ecology of human disease* 189–215.

[3] Faust, *Amebiasis* 58.

[4] *Ibid.* 28. About 50 per cent of all typhoid cases occur in the age group 15–25 years; Gay, *Typhoid fever* 13.

diseases and perhaps their virulence as they passed rapidly from one infected colonist to another (via the feces and ingestion).<sup>[1]</sup>

The role of typhoid and dysentery may be further clarified by identifying the disease agents, their introduction and the sources of human exposure. The introduction of these diseases is not problematic. The colony contained numerous carriers of both diseases. According to modern laboratory diagnostic surveys, *E. histolytica* is carried by 40 per cent of the population in the 26–30 year age group, with a decreasing incidence away from that peak.<sup>[2]</sup> And typhoid bacilli are carried by 2–30 per cent of the general population.<sup>[3]</sup> In both diseases, carriers may be symptomless, and therefore almost impossible to detect in the absence of laboratory diagnoses. The Jamestown carriers passed millions of disease organisms in their faeces, and also through the urine in the case of typhoid. The diseases were then transmitted, in all probability, through the ingestion of a contaminated water supply.<sup>[4]</sup>

But what was the water supply, and why was it contaminated in July and August and not earlier or later? The colonists drank river water. In spring, the water was safe. With river discharge at a maximum, owing to high precipitation, low evaporation and high runoff, the fresh running water swirled around Jamestown Island and flushed disease organisms downstream. But the English, operating on a temperate European model, were unprepared for the dramatic changes in the continental climate of Virginia. The water supply became contaminated as summer set in. River discharge fell, water levels receded some 10–15 feet, and Jamestown Island became a peninsula attached to the mainland.<sup>[5]</sup> Pools of standing water and stagnant marshes rimming the mainland side of the island created a virulent

[1] Faust, *Amebiasis* 26. Modern surveys for *Endamoeba histolytica* in the southeastern United States average 11 per cent positive in the general population; but among the concentrated populations of a mental hospital and an orphanage, the positives rose to 40 and 55.5 per cent, respectively. Willard H. Wright, *Parasitism in Southeastern United States: current status of parasitic diseases*, *Public Health Reports* 70 No. 10 (1955) 966–75. Gay, *Typhoid fever* 14–5 43–5

[2] Faust, *Amebiasis* 28

[3] Gay, *Typhoid fever* 43

[4] As one expert on typhoid fever has observed, “it must be remembered that a single discharge of the bowels of a typhoid patient may contain one billion typhoid bacilli, perhaps more at times, and that 1 per cent of one billion is ten million, a number of bacilli large enough to do immense damage”. George C. Whipple, *Typhoid fever: its causation, transmission and prevention* (New York: John Wiley & Sons 1908) 49–50

[5] The normal regime of Chesapeake estuaries is described here. Discharge, however, will depart from the norm of spring highs and summer lows under atypical meteorological conditions, e.g. prolonged drought or excessive rainfall, variable evapotranspiration, variable snow-melt water. Virginia, Virginia Conservation Commission, Division of Water Resources, *Surface Water Supply of Virginia: James River Basin*, Bulletins Nos 5, 13, 17, and 25 (Charlottesville and Richmond 1944–61). As the James rose in spring and receded in summer, Jamestown occupied alternatively an island and a peninsula attached to the mainland. C. A. Browne, Reverend John Clayton and his early map of Jamestown, Virginia *William and Mary Quarterly* 2nd Series XIX (1939) 5–6; The recession in river depth is estimated from depths of the main channel at Jamestown. Percy gives 6 fathoms (36 feet) in spring; an English pilot, interrogated by the Spanish in 1611, put the depth at 3½ fathoms (21 feet) at the least. The river’s annual range is 15 feet. Alexander Brown, *The genesis of the United States* I (2 vols; Boston 1890) 519. Samuel H. Yonge, *The site of old “James Towne”, 1609–1698 . . .* (Richmond: The Hermitage Press 1907)

wetland environment for *S. typhi* and *E. histolytica*. Even more deadly was the summer contamination of river water with salt, sediment and fecal material. As fresh water discharge fell, salt water invaded some 30 miles up the James estuary from Hog Point in the spring to Jamestown by mid-summer. And along the landward moving fresh-salt boundary, sediments and organic wastes were trapped by the salt plug, particularly on the north side of the James owing to the rightward deflection of the marine incursion by the earth's rotation.<sup>[1]</sup> Percy put it succinctly: "our drink [was] cold water taken out of the River, which was at flood very salt, at low tide full of slime and filth, which was the destruction of many of our men . . .".<sup>[2]</sup> At flood tide, the colonists drank water containing salinity concentrations of over 5 parts per thousand—far above the recommended standard for constant daily usage of 1 part per thousand. The colonists suffered from salt poisoning, with its characteristic symptoms of "swellings" (edema), lassitude and irritability. The idle, lazy and factious behaviour of early Virginians was, in part, the result of a steady summer diet of salty water.<sup>[3]</sup> The ebb tide, though less saline, was very turbid, organically polluted and deadly. The trapped pathogens of typhoid and dysentery, thus, floated back and forth past Jamestown with summer tide. The danger from contaminated water passed in September. River discharge increased, pushing the salt incursion and its deadly associates downstream toward Hog Point.

The 1607 epidemic of typhoid and dysentery was the first of many summer epidemics in early Virginia. Fevers, fluxes, sickness and death visited the colony recurrently between 1607 and 1624. One decisive factor underlying these repeated epidemics is the limited immunity conferred by the diseases themselves. Dysentery survivors acquire no immunity to subsequent attacks. Severe dysentery attacks invoke a limited antibody response for 2 weeks after the infection, but thereafter the survivor is susceptible to infection.<sup>[4]</sup> Typhoid attacks confer somewhat more immunity. Typhoid recurrence is usually put at 0.75–4.2 per cent; however, the recurrence rate rises to between 8 and 15 per cent in especially virulent and massive infections, like those in Jamestown.<sup>[5]</sup> Thus, survivors of dysentery and typhoid epidemics at Jamestown were only slightly less susceptible to these diseases than were newly arrived immigrants. Survivors of a Virginia summer became "seasoned" to a new disease environment; however, they were not particularly immune to future epidemics of typhoid, dysentery or salt poisoning. These epidemics recurred for one other reason—the annual summer invasion of salt water up the James, thereby contaminating the Jamestown water supply.

[1] An excellent survey of the James is Maynard M. Nichols, *Sediments of the James River Estuary, Virginia Geological Society of America, Memoir 133* (1972) 169–212.

[2] "Observations of Master George Percy" 21–2.

[3] Drinking water preferably should contain not more than 0.5 parts per thousand salt content; however, some contemporary municipal water supplies use 2 parts per thousand without public complaint. Thomas R. Camp and Robert L. Meserve, *Water and its impurities*, 2nd ed. (Stroudsburg, Pennsylvania: Dowden, Hutchinson & Ross, Inc. 1974) 2; Keitel, *Pathophysiology and treatment of body fluid disturbances* 162–4, 209–10. J. H. Bland, "Clinical physiology and four avenues of loss and gain," 133–64. A composite of early Virginian behaviour would include irritability, short-temper, factiousness and hyperbolic perceptions. The extremity of their situation accounts for some of these behaviours, salt poisoning accounts for them all. On idleness in early Virginia, see E. S. Morgan, "The labor problem at Jamestown, 1607–18" 595–611.

[4] Faust, *Amebiasis* 30–2.

[5] Gay, *Typhoid fever* 148.

This close relationship between environment, disease, and mortality in 1607 Jamestown may be stated more generally for all Chesapeake estuaries. For our purposes, an estuary is an ecological unit wherein fresh water from the land is mixed with encroaching water from the sea, producing several salinity zones: (1) the zone of fresh water, with salinity less than 0.5 parts per thousand; (2) the zone of fresh-salt transition (the oligohaline), with salinities of 0.5–3 parts per thousand; and (3) the zone of salty water, with salinity above 3 parts per thousand (includes the mesohaline, polyhaline and marine).<sup>[1]</sup> Sediment and fecal material entering an estuary are flushed out of its fresh-water portion, temporarily trapped or plugged up by the salt incursion in the oligohaline, and a large portion is eventually flushed downstream into the saltier water. Thus, pathogenic river-borne organisms are least in the fresh-water zone, maximum in the oligohaline zone, and intermediate in the mesohaline and polyhaline zones near the estuary mouth. Contamination also varies by bank side. Left bank contamination exceeds right bank owing to the deflection of the salt incursion by the earth's rotation. This geographic distribution of estuarine contamination is, in turn, directly correlated with human exposure, infection and mortality from the pathogens of typhoid and dysentery.<sup>[2]</sup> Mortality also varies seasonally with the migrations of the salt incursion. In the oligohaline zone, the probability of infection increases when the salt-fresh boundary passes by; clinical symptoms and mortality lag behind during the prepatent period, with a normal lag of about 1 week to 1 month. The location of this deadly boundary zone migrates with river discharge. In the Chesapeake estuaries, low discharge usually occurs in summer, and the salt water invades up-estuary to its landward maximum; on the James, it migrates 30 miles to the vicinity of Jamestown, where mortality rates should peak in July and August. Highest discharge customarily comes in spring and pushes the salty water to its seaward maximum; on the James, the retreat is to Hog Point where mortality should peak in April and May. Within the salty water, mortality should rise slightly in spring because of the proximity of the salt trap; however, this zone receives tidal flows of fecal material throughout

[1] Chesapeake estuaries are moderately stratified, i.e. turbulence by tidal action mixes underlying salty water and over-riding fresh water, thus bringing salt to the water surface. The variety of estuaries is discussed in a massive compendium, George H. Lauff (Ed.), *Estuaries*, Publication No. 83, American Association for the Advancement of Science (Baltimore: The Horn-Shafer Company 1967). Several articles therein are pertinent: Donald W. Pritchard, What is an estuary: physical viewpoint, 3–5; and his Observation of circulation in coastal plain estuaries, 37–44; M. M. Nichols and R. L. Ellison, Sedimentary patterns of microfauna in a coastal plain estuary, 283–8; and J. L. McHugh, Estuarine Nekton, 581–620, which contains the salinity classification. Also see D. W. Pritchard, Salinity distribution and circulation in the Chesapeake Bay Estuaries system *Journal of Marine Resources* XI (1952) 106–23

[2] The distribution of disease organisms within an estuary depends on their point of entry, the circulation and flushing time of the estuary, and the life expectancy of the disease organisms. Laboratory experiments show that coliform bacteria, an indicator of disease contaminants, die off rapidly to one-tenth their original population in a period of 0.5 to 2–3 days. The extent of downstream contamination increases when river circulation is rapid and the pollutants are flushed downstream before death. Bostwick H. Ketchum, Distribution of coliform bacteria and other pollutants in tidal estuaries *Sewage and industrial wastes* XXVII (1955) 1288–96; Clarence J. Velz, *Applied stream sanitation* (New York: Wiley-Interscience 1970) 339–79; *Wastes management concepts for the coastal zone: requirements for research and investigation*, Committee on Oceanography and Committee on Ocean Engineering, National Academy of Engineering (Washington, D.C. 1970)



the summer, thus assuring summer sickness and death. Recurrent epidemics were possible when the population occupied the fresh-salt and salt water zones.<sup>[1]</sup>

Having forwarded a geographic model of mortality, I hastily remind the reader of its crudity. The model coarsely sub-divides estuaries into three salinity zones, and hypothesizes their variable mortality. A more refined model might specify the precise concentrations of contaminants, as a function of estuarine flushing and transport and the life expectancies of pathogens, as well as the expected levels of infection and mortality. The hydrologic information for early Virginia hardly warrants such refinements.

The ample records that survive for early Virginia afford several opportunities of testing the geographic model of mortality. For the period 1607–24, deaths may be calculated from contemporary statistics of population and immigration, though these must be used with caution. Figures can lie, and early Virginians regularly juggled population estimates to suit their purposes. The most probable causes of death are deduced from colonists' descriptions of the timing and symptoms of disease and the plausibility of their explanations as to the causes of death. The locations of population and of mortality derive from contemporary accounts. Particularly useful is the geographic census of the living and dead for 1623–4.

The first question at issue is the relationship of mortality and the location of population in early Virginia. A chronological survey of the period 1608–24 reveals the recurrent deadliness of Jamestown summers. When population was concentrated in the town, mortality rates invariably rose above 30 per cent; and when the population dispersed, death rates declined sharply.

The first two summers in Virginia were disastrous; the third offered the first glimmer of hope. As of October 1608 perhaps 244 colonists had come to Jamestown, and 144 of them were dead.<sup>[2]</sup> But the toll of death fell abruptly between October 1608 and the summer of 1609, when by the most liberal estimates just 21 of 130 persons died, including 11 by drowning. This anomaly of survival deserves comment.<sup>[3]</sup> Captain John Smith claimed credit for this success, and rightly so.

[1] The expected timing and location of disease morbidity and mortality rest on the assumption of "average" climatic conditions and "normal" estuarine circulation, i.e. peak discharge and salt retreat in spring, and low discharge and salt incursion in late summer. Atypical weather conditions could alter the timing and location of disease incidence. The timing of epidemics is affected also by physiological factors. The increased incidence of typhoid and dysentery in late summer may have to do with increased human output of pathogens at that time. Counts of coliform bacteria in the Detroit River rose steadily in spring, reaching a peak in August; the reasons underlying this increased productivity are incompletely understood. For our purposes, this increased summer output should produce higher mortality on the landward edge of the oligohaline than on the seaward edge. Velz, *Applied stream sanitation* 239–42.

[2] In December 1606 144 colonists went to Virginia; 104 were left by Newport in June 1607; 38–40 survived in January 1608; 100–120 immigrants arrived between January and September 1608; and 60 were alive in October 1608. The mortality rate in the text is from Hecht, "The Virginia colony" 68 and Brown, *The first Republic* 55, 58–9, 68. Brown's population and immigration figures are usually accurate, and I rely on them frequently. However, his friendliness toward the Sandys administration from 1619 to 1624 and its "democratic" character leads him to minimize the mortality problem then, while he is excessively critical of mortality under the Crown and under Thomas Smythe.

[3] Friends of Smith claimed that only 7 or 8 died out of 200. Brown shows 130 alive in October 1608; 11 drowned in mid-January, and not more than 109 survived. Arber, *Travels and works* I, 154–7. Brown, *The first Republic* 70–1. Philip L. Barbour, *The Jamestown voyages under the first charter, 1606–1609 II Hakluyt Society* 2nd Series, CXXXVII (2 vols; Cambridge: published for the Hakluyt Society at the University Press 1969) 411.

Smith, though a vainglorious man, was also a sensitive ethnographer.<sup>[1]</sup> He carefully recorded the Indians' seminomadic economy, and in all probability he understood the adaptive value of Indian movements. In the spring, the Indians congregated along the James estuary, subsisting on marine life while they planted their crops of corn, pumpkins, beans and so forth. As summer approached, the tribes dispersed into smaller groups, residing usually on a hill with a freshwater spring, yet near the river where they gathered fish, oysters and crabs. By dispersing, the Indian bands avoided the unhealthy estuarine zone while exploiting scattered edible plants and animals during this leanest of seasons.<sup>[2]</sup> But dispersal toward healthier sites had its price. The scattered bands were politically and militarily weak. They sniped at their vulnerable, sick and weak English enemies, but a summer war of attrition was impossible. As this flux in Indian power eluded most Virginians, they were terrified by late summer. Percy fully expected annihilation in 1607, and he marvelled that God had saved them by putting "a terrour in the Savages hearts".<sup>[3]</sup> Smith saw things more clearly; the dispersed and fragmented Indians were almost as vulnerable as the whites. In 1609, he dispersed his men with impunity. Smith also understood Indian generosity in the fall. Then the dispersed Indians reassembled, harvested their crops and gorged themselves. Their

[1] The full story of Smith's ecological and ethnographic sensitivity and his application of this knowledge remains untold. Arber, *Travels and works* I, 61-70; Maurice A. Mook Virginia ethnology from an early relation *William and Mary Quarterly* 2nd Series XXIII (1943) 101-29. Philip L. Barbour, *The three worlds of Captain John Smith* (Boston: Houghton Mifflin Company 1964) 243-76. Smith can be eulogized too much. While others died in Jamestown, he explored the healthier reaches of the Chesapeake. In 1608, Smith returned to Jamestown long enough to see the summer sickness, and then he was off again. Brown, *The first Republic* 60

[2] For the Indians, the zone of the fresh-salt transition was assuredly unhealthy and perhaps as deadly for them as it was for the English. The Indians, had they remained in this zone during the summer, would have consumed salty and slimy (sediment) river water; instead, they scattered in small bands, locating themselves on hill sites with good spring water. Perhaps, too, Indian dispersal avoided water-borne disease. Although some scholars maintain that typhoid and dysentery were not present in America before 1492—the evidence, however, is not overwhelming on this score—these diseases could have spread into the Virginia tidewater during the sixteenth century owing to European contact. In pre-settlement New England, for instance, very slight European contact with the Indians contributed to a massive epidemic of plague in 1617. Similarly, the Virginia tribes could have been infected by tribes in the vicinity of the Roanoke colony during the 1580's or possibly by the lost colonists, whom legend has it lived among the Indians. One student of Indian disease believes that dysentery was widespread among the North American woodland Indians before the permanent settlement of the English and French. In sum, the oligohaline was unhealthy and in all probability deadly for the Indian population of Virginia. Woodrow Borah, *Hispania Victrix; American Debellata: the demographic meaning of conquest*, *Revista de Historia* (São Paulo), in press, as summarized and cited in William M. Denevan (Ed.), *The native population of the Americas in 1492* (Madison and London: University of Wisconsin Press 1976) 5; Whipple, *Typhoid fever* 117-8; Sherburne F. Cook The significance of disease in the extinction of the New England Indians *Human Biology*, XLV (1973) 485-508; Colonel P. M. Ashburn, pp. 158-9 of Frank D. Ashburn (Ed.), *The ranks of death: a medical history of America* (New York: Coward-McCann, Inc., 1947); Alfred W. Crosby, Jr, *The Columbian exchange: biological and cultural consequences of 1492*, *Contributions in American Studies*, 2 (Westport, Connecticut: Greenwood Publishing Company 1972). More recently, researchers on the James River have discovered a new water-borne bacterial disease called the "coastal disease" which is a potentially fatal disease of the lungs. *The Baltimore Sun*, 25 October, 1977, A8

[3] "Observations of Master George Percy" 22

full bellies made them charitable, and they brought "Bread, Corne, Fish and Flesh in great plentie" to the confounded colonists.<sup>[1]</sup> With the onset of winter, the Indians once again fragmented into small bands and migrated upland into their piedmont hunting grounds, where they stalked deer, bear and other game animals. Smith's genius was in placing the puzzling Indian behaviour and subsistence strategies into a coherent ecological whole. He comprehended that the colony's survival depended on emulating the Indians' seminomadism, at least during the deadly summer season.<sup>[2]</sup>

In late May 1609, President Smith scattered the Jamestown settlers into the surrounding countryside. His scheme infuriated Captain Gabriel Archer, who described more than he understood: "Howbeit when Captaine Argoll came in [about July 10, 1609], they were in such distresse, for many were dispersed in the Sauages townes, living vpon their almes for an ounce of Copper a day; and four score lived twenty miles from the fort and fed vpon nothing but oysters eight weekes Space . . .".<sup>[3]</sup> Smith's scheme of dispersal, though repugnant to Argall, was the wisest to date. But the scheme encountered other opposition in August with the arrival of 185–270 immigrants. Smith was able to dispatch one-third of the colonists to Nansemond on the south side of the river in the salt water and another one-third to the fresh water at the Falls near the head of the James. But the rest stayed in Jamestown, assuredly against Smith's better judgement. Predictably, sickness ravaged 100 Jamestown colonists, and 50 died by October. Yet at Nansemond in the salt and at the Falls in the fresh, few sickened and none died.<sup>[4]</sup> Indian behaviour had given Smith the key to life in the James estuary, but this precious knowledge was soon lost. He was relieved of the presidency in October and returned to England; with him went the schemes of seminomadism and summer dispersal. The colony once again clustered at Jamestown, and death hung heavy over the settlement.

Between Smith's departure and Gates' arrival in May 1610, the colony experienced the infamous "starving time". The accounts of hundreds starving, of cannibalism, and other inhumanities, have proven irresistible. But these accounts are biased, sensationalized and exaggerated. They have warped the death toll and its causes out of all proportion and have diverted attention from the summer epidemics. In the first place, the death toll in the winter of 1609–10 was much less than is usually assumed. The most common error has been the belief that 490–500 immigrants came to Virginia in October, just 50–60 survived when Gates

[1] "Observations of Master George Percy" 22

[2] The conclusion that Smith fully appreciated Indian ecology and its seasonal avoidance of unhealthy areas seems, to me at any rate, inescapable. Smith's careful ethnographic descriptions of the Indians preceded his accession to the colony's leadership and his decision sending the colonists out to live in scattered bands among the Indians. The Captain knew that dispersal was an extraordinary measure because it directly contravened the Company's instructions insisting on a clustered settlement on a defensible site, preferably an island. For background see Alden T. Vaughan, *American genesis: Captain John Smith and the founding of Virginia* (Boston and Toronto: Little, Brown and Company 1975)

[3] Arber, *Travels and works* I xcvi

[4] Staying in Jamestown during August was inconsistent with Smith's strategy, and so I conclude that his opponents were responsible for the return to the town. By summer's end, 1609, the population stood at 250. *A voyage to Virginia in 1609: two narratives Strachey's "True Reportory" and Jourdain's "Discovery of the Bermudas"*, Louis B. Wright (Ed.) (Charlottesville: University Press of Virginia 1964) 83

arrived in May 1610, hence over 400 died.<sup>[1]</sup> In fact, the Virginia population in October stood at 250 or less; and after Smith departed with 30 unruly youths, 220 colonists remained. At least 15 of these were killed by Indians, and 25–30 others returned to England, leaving 180 in the colony.<sup>[2]</sup> When Gates arrived in May, he found 40 men in good health, along with President Percy, at Point Comfort near the mouth of the James. And at Jamestown, 60 ragged men dragged out to meet Gates.<sup>[3]</sup> In other words, 100 survived the winter, 15 were killed by the Indians and 80 died from other causes.

Was starvation the cause of death? Enemies of the Company and Sir Thomas Smythe's administration placed the blame on starvation resulting from inadequate provisions. Purportedly, "famine compelled us wholly to devour those Hogs, Dogges & horses that weare then in the Collony . . ." along with vermin and human flesh.<sup>[4]</sup> Yet there are serious inconsistencies surrounding the starving time. Gates reported that 600 hogs were destroyed, which at conservative dressweights of 50 pounds per hog amounted to 30,000 pounds for 200 colonists or less—or about 150 pounds per capita during the seven months.<sup>[5]</sup> And supplemented by 500 chickens, dogs, 7 horses, rats, snakes and other vermin, the colonists' diet seems sufficient to ward off starvation—even without human flesh.<sup>[6]</sup> Gates offered a different interpretation. He noted that Powhatan stepped up hostilities, confining the colonists to Jamestown between October and May. Some of the colonists were murdered, others fled, "and most by drinking of the brackish water of James Fort weakened and endangered, famine and sickness by all these means increased".<sup>[7]</sup> Brackish water, probably contaminated with typhoid and dysentery, is implicated once again, but this time in winter. One source of salty water, of course, was shallow wells, tapping brackish aquifers contaminated by pathogens percolated downward into the ground water. Another possible source of bad water was the river, contaminated during the severely cold winter of 1609–10. Climatologists have observed that cold temperatures and subsiding air depress rainfall. A cold, dry

[1] The starving time is embedded in Virginia's historical lore, embracing everything from AAA guide books to Edmund Morgan's sophisticated research. Yet another and less dramatic explanation may be suggested. Proponents of the starving time err in assuming that 490–500 immigrants reached Virginia before the winter of 1609–10. However, only 185–270 arrived, finding about 109 survivors, making a total of 294–379. Three hundred seems about right, as it fits Strachey's estimate. Fifty of these died at Jamestown in August 1609. Brown, *The first Republic* 97, 109, 112–3. One hundred were alive when Gates arrived in May 1610. *A voyage to Virginia* 82–3, 115

[2] Brown, *The first Republic* 97, 109, 112–3; Despite his careful analysis of mortality, Brown is anxious to blame Captain Smith and the Company and so he accepts the thesis of starvation promulgated in "A Trewe Relaycon: Virginia from 1609 to 1612" Tyler's *Quarterly Historical and Genealogical Magazine* III (1922) 264–70; A Brief Declaration of the Plantation of Virginia . . . in 1624, *Colonial Records of Virginia* (Richmond: R. F. Walker, Superintendent of Public Printing 1874) 70–3; William Stith, *The History and the First Discovery and Settlement of Virginia*, Introduction by Darrett B. Rutman (New York: Johnson Reprint Corporation 1969) 108–17

[3] Percy's abandonment of Jamestown for the healthier Point Comfort site was pragmatic, but not the most heroic of gestures. *A voyage to Virginia* 62–3

[4] "A brief declaration" 71

[5] Gates' comments are excerpted in *A Voyage to Virginia*, 99; also, Strachey's remarks, *ibid.* 86–7

[6] Gates' hog estimate was confirmed by Smith. *A voyage to Virginia*, 86–7. Arber, *Travels and works* I 167

[7] The tragedy was also blamed on "idleness", perhaps the result of salt intoxication. *A voyage to Virginia* 98–9



winter, common in many parts of the mid-latitudes during the late fifteenth and early sixteenth centuries, would have lowered river discharge, and delayed the retreat of the estuarine salt incursion, fecal material and sediment from Jamestown.<sup>[1]</sup>

We cannot say conclusively that typhoid, dysentery and salt poisoning were the principal causes of death in that winter; however, we can suggest that the case for massive starvation is far from proven. For instance, the mortality rate of 44.4 per cent is much lower than the rate usually suggested by proponents of the "starving time". Starvation appears dubious given the livestock available and consumed in the winter. Moreover, the mortality rate is very similar to expected and observed rates of death from typhoid and dysentery for 1607 and 1608. Finally, winter mortality in early Virginia was rare except in extremely severe winters (e.g. 1607-8 and 1609-10).<sup>[2]</sup> Cold, dry winters and estuarine hydraulics could have produced a contaminated water supply and epidemic typhoid and dysentery in the so-called "starving time".

With the arrival of Lord de la Warr in June 1610, Jamestown was retained as the colony centre. Three hundred and fifty were alive in mid-June, the sickness began 1 month later, and 150 (43 per cent) had died by the end of summer. Fifty more died by April 1611.<sup>[3]</sup> Colony leaders strongly suspected the Jamestown water supply as the cause of death. Gates and Strachey stated as much, and Gates and de la Warr, on their return to England in the fall of 1610, communicated their fears to the Company.<sup>[4]</sup> Jamestown's days were numbered, or so it seemed.

The establishment of a healthier town site took time, and meanwhile summer death continued. Thomas Dale arrived in Virginia on 22 May 1611 with 300 colonists, bringing the colony strength to 480.<sup>[5]</sup> By mid-June, Dale had chosen

- [1] For seventeenth-century climate, see H. H. Lamb, *The history of our climate: Wales*, in *Climatic change with special reference to Wales and its agriculture*, James Taylor (Ed.), Memorandum No. 8 (1965), University College of Wales, Aberystwyth, 1-18; C. E. P. Brooks, *Climate through the ages: a study of the climatic factors and their variations*, 2nd revised edition (New York: Dover Publications, Inc. 1970) 359-78; The extremely cold winter of 1609-10 is noted in Brown, *The first Republic* 113. Salty water at Jamestown in 1609-10 would explain the presence of water too cold to wade in for oysters, yet unfrozen because the salt incursion lowered the freezing point. "A brief declaration" 71. Twentieth-century records of the James River provide evidence of winter discharge falling below late summer levels. However, salinity records are too recent and spotty to indicate a winter salt incursion. Virginia Conservation Commission *Surface water supply* Nos. 5, 13, 17 and 25
- [2] Arber, *Travels and works* I 23, 98; Brown estimates that 57 of 110 colonists died between January and April 1608 and that 25 of 83 died between April and October 1608. The winter mortality of 52 per cent exceeds slightly the 44 per cent rate (80/180) for the cold winter of 1609-10. Brown, *The first Republic* 57. Arber, *Travels and works* II, 398, 407, 434
- [3] Gates withdrew from Jamestown with 200 in June 1610. The 40 others at Point Comfort probably were not included in this count. The colony thus numbered 200-240. De la Warr arrived with 150 men, putting the colony at 350-390. The colonists were heartened when De la Warr announced he had provisions for 400 men for 1 year. They would have been less cheerful if De la Warr had brought 200-300 immigrants, as is sometimes asserted. *A voyage to Virginia* 85, 115. Brown, *The first Republic* 116, 128, 134-9. Richard L. Morton, *Colonial Virginia* I (2 vols; Chapel Hill: University of North Carolina Press 1960) 27-8; Hecht, *The Virginia colony* 330; "A Trewe Relaycon": Virginia from 1609 to 1612" 269-70
- [4] For Strachey's insights, *A voyage to Virginia* 82-3. Morton, *Colonial Virginia* I, 28-9
- [5] One hundred and fifty were alive in March 1610; 30 arrived soon after, and Dale brought 300 in May 1611—a total of 480. Brown, *The first Republic* 138-9, 149. Ralph Hamor, *A true discourse of the present state of Virginia*, introduction by A. L. Rowse (Richmond: The Virginia State Library 1957) 26

a new town site—the Falls at the head of the James—but building did not commence until September. The colonists spent the summer in Jamestown, and the sickness began in early July. A few days later Dale instituted martial law. But tough discipline did not thwart disease. At least 240 of the colonists became so sick they could not work.<sup>[1]</sup> A death toll of about one-third of the population, or 160, would be consistent with summer mortality and with later population estimates. At summer's end in 1611, the colony population stood as follows: Dale's 320 survivors, plus 300 immigrants brought by Gates in August, all of whom were evacuated to the healthier Falls site, and 62 brought by Argall in late September putting the colony total at 682—a figure just slightly below the 700 estimated for early 1612 by a Spanish prisoner at Jamestown.<sup>[2]</sup>

Rome was not built in a day, nor was Henrico, the new town at the Falls. Construction began in autumn, 1611, and continued through winter. But the schedule was interrupted by spring planting in 1612, and full-scale settlement of the new town awaited the end of the harvest. My guess is that the majority of colonists spent the summer at Jamestown. The mortality results suggest as much. According to the Spanish prisoner Molina, 350 died out of a total population of 700. Molina's report appears accurate. Seven hundred colonists seems about right for spring 1612. Molina, however, gives two estimates of the survivors in May 1613—either 305 or 350. The death rate in 1612–13 was probably 50 per cent or more.<sup>[3]</sup>

With the establishment of Henrico and the general dispersal of population between 1613 and 1616, early Virginia enjoyed its healthiest era. By 1614, Jamestown had dwindled as the colony's centre, and the population shifted toward the head of the James River. Rolfe's description of settlement in 1616 revealed that Jamestown contained just 19 per cent of the colony population, and just 32.3 per cent resided in both the oligohaline (Dale's Gift and Jamestown) and the salt water (Kecoughtan). The remaining 67.7 per cent occupied the fresh-water zone at Henrico, Bermuda Nether Hundred and West Sherly Hundred.<sup>[4]</sup> Mortality was rarely mentioned in the contemporary correspondence or accounts of these years, and for good reason. The population in May 1613 consisted of 305–350 persons, and by May 1616, 45 immigrants had arrived, and 351 colonists survived.<sup>[5]</sup> The

[1] Brown, *The first Republic* 149–55; Morton, *Colonial Virginia* I, 28–31; Brown, *Genesis* I, 506–7; Darrett B. Rutman, *The Virginia Company and its military regime* pp. 1–20 of *The Old Dominion: essays for Thomas Perkins Abernathy* (Charlottesville: The University Press of Virginia 1964)

[2] Brown, *The first Republic* 156, 172; Arber, *Travels and works* II, 509; "Letter of Diego de Molina, 1613 [May]" *Narratives of Early Virginia* 220, 223–4

[3] As late as May 1613, Jamestown contained almost one-half of the colony's population, and Henrico only one-third. The pattern surely changed by the summer of 1613. "Letter of Diego de Molina, 1613" 223–4. Hamor, *A true discourse* 32. Dale's letter of June 1614 is enlightening. Although his obligation in Virginia was complete, he believed the colony was in "desperate hazard". Abandoning her might reflect on his reputation. Perhaps too, Dale had to endure another summer to see if the healthy year preceding (1613) resulted from his settlement policies or from dame fortune. *Ibid.* 51–9

[4] John Rolfe, *A true relation of the state of Virginia left by Sir Thomas Dale Knight in May last 1616* (New Haven: Yale University Press 1951) 33–41; Hamor, *A true discourse* 26–33; Charles E. Hatch, Jr, *The first seventeen years: Virginia, 1607–1624*, Jamestown 350th Anniversary Historical Booklets, No. 6 (Williamsburg: The Virginia 350th Anniversary Celebration Corporation 1957), 32–3

[5] Brown, *The first Republic* 220, 224, 229. Hecht, "The Virginia colony" 332. Writing in 1614 about the martial law invoked in 1611, Hamor defended Dale's measures, "for more deserued death in those daies, then do now the least punishment". Healthful conditions had marvelously reformed idle and factious Virginians! Hamor, *A true discourse* 27

mortality rate had declined sharply. Assuming no natural increase, the Virginia population either held steady or declined at a rate of about 15 deaths per year—an astonishing annual mortality rate of about 3·8 per cent.

The marked improvement in mortality rates following the redistribution of population into healthy fresh-water environments is consistent with the model used here; however, proponents of a "seasoning" thesis have maintained that a simultaneous reduction in immigration may have reduced death rates. They suggest that mortality rates among immigrants were very high, but fell sharply among the survivors, who were then seasoned to the Virginia disease environment. The seasoned survivors were less susceptible (immune) to disease in future years. An alternative hypothesis, and the one favoured here, maintains that seasoned colonists were nearly as susceptible as newcomers to typhoid, dysentery and salt poisoning; and their vulnerability can be demonstrated for the period under discussion. Immigration to Virginia came to a virtual standstill in the summer of 1611. The seasoning thesis would posit a sequence of high mortality rates in that summer, the survival of seasoned colonists and a sharp mortality reduction in the summer of 1612. In fact, we have shown that mortality remained high in both summers. Death rates dropped dramatically in 1613, after the Virginians shifted their settlements into the fresh water. Environment and location were the decisive factors lowering mortality between 1613 and 1616. Immigration and seasoning were largely irrelevant.<sup>[1]</sup>

The healthy era, 1613–16, was the product of a lengthy and painful process of environmental learning and adjustment. The three years from 1607 to 1610 were spent enduring death and identifying its geographic pattern and its causes. By the spring of 1610, colonist leaders had associated death, water supply and the Jamestown environment. They persuaded the Company in London by the fall of that year. Implementation of a new settlement distribution consumed the next 3 years from the winter of 1610–11 to the fall of 1613. During these years, Dale reconnoitred, chose a site, began construction of Henrico, cleared land for crops and instituted an aggressive Indian campaign. Thus in 7 years, the Company had perceived the solution to summer mortality, and Dale worked swiftly toward that end. But the re-settlement scheme was greatly facilitated by the Indians. Dale's provocative encroachment into Powhatan's territory at the head of the James met little resistance from the chief. Perhaps Powhatan's advanced age and the capture of his daughter Pocohontas tempered his retaliation, but his mysterious behaviour suggests intrigue. The chief remained incommunicado from May 1613 to March 1614 and he removed his quarters from the James to the Pamunky River. Is it not possible that Powhatan had his hands full with the hostile Monacans on

[1] Seasoning was at once a well-recognized process in Virginia and a theory of curative medicine, i.e. treatment of individuals. By exposing the individual to infection in a new disease environment, future susceptibility was reduced. The theory worked fine for self-immunizing or debilitating diseases such as malaria, bacillary dysentery and paratyphoid. But curative medicine worked miserably on non-immunizing, virulent diseases. Exposure to these diseases brought death year after year. The only effective remedy against them, at least before vaccines and antibiotics, was the preventive medicine of environmental modification or avoidance. Since Virginians were powerless to change the oligohaline, the best course was to avoid that zone. An excellent discussion of the seasoning process and the role of malaria is Darrett B. Rutman and Anita H. Rutman, *Of agues and fevers: malaria in the early Chesapeake* *William and Mary Quarterly* 3rd Series XXXIII (1976) 31–60; Blanton, *Medicine in Virginia* 37–41; Ashburn, *The ranks of death* 118–23, 159–60; Duffy, *Epidemics in Colonial America* 214–8; May, *The ecology of human disease* 26

his western flank, and until they were subdued, he temporarily conceded Dale the James River head? Whatever Powhatan's motives, the English colony profited from his passivity.<sup>[1]</sup>

When Dale left Virginia in the spring of 1616, he felt confident that the colony would endure. The mortality problem had been solved by diminishing Jamestown's importance and locating the settlements in healthier zones. With sickness and death on the wane, the healthy colonists produced a surplus of food. Trade relationships altered. Formerly, the colonists begged, stole or traded for Indian food; now, the Indians came seeking the colony's corn.<sup>[2]</sup> Healthy conditions continued through the summer of 1616. There were no reports of widespread mortality, and the colony probably contained 335–351 colonists. But with the arrival of a new governor in the spring of 1617, all of Dale's insights were abandoned, to be painfully relearned.

Governor Samuel Argall was not one to learn from his mistakes. This was the same Argall who earlier had condemned Smith's dispersal of colonists in the summer of 1609. As governor in the spring of 1617, Argall was again appalled by the state of the colony and Jamestown where: "he found but five or six houses, the Church downe, the Palizado's broken, the Bridge in pieces, the Well of fresh water spoiled; the Store-house they used for the Church; the market-place, and streets, and all other spare places planted with Tobacco: . . . the Colonie dispersed all about, Planting Tobacco".<sup>[3]</sup> On 9 June Argall wrote to the Company that he "likes James Town better than Bermudas 40 miles aboue it; and will strengthen it".<sup>[4]</sup> Argall must have succeeded in realigning settlement, for that summer a great mortality ensued. Death struck 105–115 of the 415 colonists, and suddenly the mortality rate had risen from nil to 25 per cent.<sup>[5]</sup>

The realignment of settlement begun by Argall and continued under the Sandys administration was one of the principal causes of death until 1624. The hard-won knowledge of the environment and the adjustments made between 1607 and 1617 were abandoned. Between 1617 and 1623, 36 new settlements dotted the James estuary, and 13 of them occupied the oligohaline and the saltier water.<sup>[6]</sup> More importantly, population shifted into the lower estuary, and Jamestown was reaffirmed as chief city and centre of government. The extent of realignment is

[1] Hamor, *A true discourse* 54–5; William Strachey, *The historie of travell into Virginia Britania* (Louis B. Wright and Virginia Freund, Eds.) 2nd Series, No. 103 (Hakluyt Society 1953) 105–6; Nancy Oestrich Lurie, *Indian cultural adjustment to European civilization* pp. 33–60 of *Seventeenth-century America*, (Chapel Hill: University of North Carolina Press 1959). James Morton Smith (Ed.); Wesley Frank Craven, *White, red, and black: the seventeenth-century Virginian* (Charlottesville: The University Press of Virginia 1971) 46–50

[2] Rolfe, *A true relation* 36

[3] Arber, *Travels and works* II, 535–6

[4] Susan Myra Kingsbury (Ed.), *The records of the Virginia Company of London III* (4 vols; Washington, D.C.: Government Printing Office 1933) 73

[5] *Ibid.* 92. The mortality rate is based on the following: probably 335 (my estimate) were alive in May 1617, plus 80 brought in by Argall, or a total of 415. In May 1618 the colony contained about 400, of whom 90–100 had arrived between March and May 1618. Subtracting these from the 400 yields 300–310 alive in March 1618. Thus, from May 1617 to March 1618, 105–115 had died. Arber, *Travels and works* II, 535–6; Brown, *The first Republic* 253–6, 260, 277; Hecht, *The Virginia Colony* 333–4; Evarts B. Greene and Virginia D. Harrington, *American population Before the Federal Census of 1790* (New York: Columbia University Press 1932) 135

[6] Hecht "The Virginia Colony" 174, 361–3



revealed in two sources: the census of 1623–24 supplemented by the deaths from the massacre of March 1622 which help provide a more accurate picture of population distribution from 1618 to 1622 (Table 1).<sup>[1]</sup> Seventy-one per cent of the Virginia colonists resided in the oligohaline and salt water zones; 28 per cent occupied the fresh water—almost a direct reversal of the pattern under Dale, when 68 per cent lived in the fresh-water zone.

TABLE 1  
*Population distribution in 1622–4 (and the probable distribution from 1617 to 1624)*

Estuarine Zone	Massacre deaths	Living 1623–4	Dead 1623–4	Total	% of Population
Fresh water	209	289	57	555	28.5
Oligohaline	145	603	211	959	49.3
Mesohaline and polyhaline	0	303	101	431	22.2

*Note:* Does not include population on the Eastern shore nor on the recently arrived vessels.

Increased mortality accompanied the shift in population. Several thousand colonists died between 1618 and 1624, and disease was an important cause. Comments on summer sickness and death increasingly punctuated colonial correspondence. But disease was not the sole killer. Indian attacks, starvation and plague also contributed. While the surviving evidence precludes a precise bill of mortality, some estimates of disease-related deaths can be made from the census of 1623–4.

As a benchmark year 1623–4 is here used for estimating the usual 1618–24 mortality rate from typhoid, dysentery and salt poisoning. Several bits of evidence suggest these diseases as the principal causes of 1623–4 death: (1) the reports of summer sickness and death in that year; (2) the absence of other reported causes of mortality; (3) the abundant food supply, making starvation an unlikely cause of death; and (4) the census listing of colonists killed, presumably by the Indians, permits the exclusion of these deaths from our disease estimate.<sup>[2]</sup> Typhoid and dysentery are also implicated by the spatial pattern of death recorded in the census of 1623–4. Among those settlements reporting deaths during the year, 16.7 per cent died in the fresh-water zone; 37.1 per cent in the oligohaline; and 23.3 per cent in the saltier portion of the James estuary (Table 2).<sup>[3]</sup> The match between reality and our estuarine model is good, but not perfect. Fresh-water death rates are

[1] Lists of the living & dead in Virginia, February 16, 1623 [4] *Colonial Records of Virginia* 37–60. Kingsbury *Records III*, 565–71. The census of 1623–4 alone gives a misleading impression of population distribution between 1618 and 1622. Massacre casualties on 22 March, 1622 were heaviest upriver from Jamestown, and hence I have included them as a more accurate representation of population geography under the Sandys administration

[2] Brown, *The first Republic* 569–70; Edmund S. Morgan, *Slavery and freedom: the ordeal of colonial Virginia* (New York: W. W. Norton & Co., Inc. 1975) 104–5; The year following the massacre of 1622 was very sickly, but the resultant mortality probably antedated the census of 1623–4. *Narratives of early Virginia* 438; Morton, *Colonial Virginia I*, 83–90

[3] "Lists of the living & dead" 37–60

TABLE 2  
*Estuarine zones and mortality rates, 1623-4\**

Settlements	Estuarine Zone	% Dead	
College Land	Fresh water	0.0 (0/33)	
Neck of Land		9.8 (4/41)	
West & Sherlow Hundred		17.9 (10/56)	
Jordan's Journey		16.0 (8/50)	16.7 (50/299)
Flourdieu Hundred		28.6 (18/63)	
West & Sherlow Hundred Island		17.9 (10/56)	
James Cittie within the Corporation	Oligohaline	32.7 (89/272)	
Plantation over against James River		45.8 (65/142)	
Hogg Island		8.8 (3/34)	37.1 (207/558)
Martin's Neck		51.0 (26/51)	
Warwick Squarke		40.7 (24/59)	
Elizabeth City	Mesohaline, polyhaline	23.3 (98/420)	23.2 (98/420)

\* Table includes only those settlements returning lists of dead. "Killed" colonists are not here included among the dead.

higher than expected, perhaps reflecting the severe disruptions in this area caused by the massacre of 1622. Another peculiarity is Hog Island in the oligohaline, where only 8.8 per cent died. A safer right bank location, the removal of pollutants towards the north bank by a river meander and the small population probably combined to make Hog Island a healthy micro-environment. Otherwise, the census pattern points towards death by typhoid and dysentery in the oligohaline and the salty lower James.

Having isolated these diseases as probable causes of death, we can estimate their usual contribution to Virginia mortality. The annual disease mortality rate (Table 3) is estimated as the sum of the products of the ecological zone death rates (Table 2) and the population distribution for 1618-24 (Table 1). A disease mortality rate of 28.3 per cent per year is indicated; for typhoid, dysentery and salt poisoning alone, 23.5 per cent, with the oligohaline contributing 18.3 per cent and the salt water 5.2 per cent. In the fresh water 4.8 per cent died, but the causes are not known.

TABLE 3  
*Estimated annual disease mortality rates based on population distribution and estuarine zone mortality rates 1618-24*

Estuarine zone	% of Colony population (1)	Annual mortality rate (2)	% of Total mortality rate (1 x 2)
Fresh water	28.5	16.7	4.8
Oligohaline	49.3	37.1	18.3
Mesohaline, polyhaline	22.4	23.3	5.2
Estimated annual disease mortality rate for Virginia			28.3

Argall and the Sandys administration, by redistributing Virginians into the most deadly zones, share the responsibility for the deaths of 24-28 per cent in any single year. But the gravity of their offence worsened with time. Epidemics struck year after year, killing immigrants and seasoned colonists alike. Disease claimed considerably more colonists than 28 per cent between 1618 and 1624. The overall contribution of disease to death is estimated from the several censuses and immigration figures, and the basic data are set out in Table 4.<sup>[1]</sup> Between December 1618 and February 1624; about 5,145 persons resided in or immigrated to Virginia; 24.8% survived in 1624, 49.3% died from disease and 25.9% died from other causes or went back to England. Two of every three deaths resulted from typhoid, dysentery and salt poisoning. These diseases were the principal killers in some years, and they were significant contributors in all.<sup>[2]</sup>

The Company and colony leaders tried desperately to reduce summer mortality, but failed because they misperceived its causes. Preventive measures were aimed at the immigrants and not environment and population distribution. Guest houses (hospitals) were established and immigrant arrivals were scheduled for fall after the sickly summer months, all done on the false assumption that "seasoned" colonists would survive.<sup>[3]</sup> But seasoned colonists stood little chance of survival in the oligohaline zone, as revealed in the muster of 1625. Fifty-seven settlers gave arrival dates before 1616. Twenty-four resided in the freshes, 25 in the salt, and just 9 in the oligohaline. Older settlers, those arriving before 1620 of all giving arrival dates, made up about one-fifth of the population in the oligohaline, one-third in the salt water and two-fifths in the fresh.<sup>[4]</sup> These spatial and environmental patterns of death went undetected by the Company, and that inability was instrumental in its dissolution.

The demise of the Virginia Company in 1624 signalled a new era in Virginia demography. The old constraints focusing the colony on Jamestown and the oligohaline were relaxed, and mortality fell. Between 1625 and 1634, Virginia's

[1] Population estimates for 1618-22 and 1624 are from: Brown, *The first Republic* 328-9, 375, 381, 415, 462, 464, 466-7, 503-5, 612; Morgan, *Slavery and freedom* 412-3; Greene and Harrington, *American population* 134-6; immigration estimates are from Brown *The first Republic*, see above for pages. Hecht, *The "Virginia colony"* 334-45; The unknown population of April 1623 is estimated by working backwards, i.e. population of February 1624 (1275) minus immigrants between April 1623 and February 1624 (405) plus deaths during the period (371) equals population as of April 1623 (1241); "Lists of the living & dead" 37-60

[2] These estimates of annual disease mortality permit an assessment of other causes of death. For example, Morgan has suggested starvation and malnutrition, occasioned by control of Virginia's food and labour supply by a handful of private capitalists. The most likely years for such crass behaviour raising mortality were 1620-2, when causes *other than* Indian killings and diseases contributed 40-50 per cent of all deaths. Morgan, *Slavery and freedom* 92-107. Note, however, that immigration was also heaviest in these years—suggesting Craven's point of inadequate provisioning of the immigrants by the Company. Probably both Company and private wheeler-dealers were responsible for the increased death rate; in any case, the critical years seem to have been 1620-2. Wesley Frank Craven, *The dissolution of the Virginia Company: the failure of a colonial experiment* (Gloucester, Massachusetts: Peter Smith 1964) 152-3

[3] The Company believed in the curative medicine of seasoning, rather than the preventive medicine of settlement dispersal. Craven, *Dissolution of the Virginia Company* 148-75; Kingsbury *Records III*, 275, 301-2

[4] John Camden Hotten (Ed.), *The original lists of persons of quality . . . 1600-1700* (New York: Empire State Book Co. 1931) 200-65

TABLE 4  
*Disease-related death estimates in Virginia, 1618-24*

Time period	Population at beginning	Population at end	Immigrants	Overall mortality rate*	Disease-related deaths (using estimate of 28.3% per year)	Disease death/total
December 1618-March 1620	600	887	814-914	37.3%-41.4% (527-627/1,414-1,514)	402-430 (28.3%)	402-430/527-627 (68-76%)
March 1620-March 1621	887	843	1,051	56.5% (1,095/1,938)	550 (28.3%)	550/1,095 (50.2%)
March 1621-March 1622	843	1,240	1,580	48.8% (1,183/2,423)	688 (28.3%)	688/1,183 (58.2%)
March 1622-April 1623	1,240	1,241	695	35.9% (694/1,935)	347 (17.9%)†	347/694 (50.0%)
April 1623-February 1624	1,241	1,275	405	22.5% (371/1,646)	371 (22.5%)‡	371/371 (100.0%)

*Summary:* Population in February 1624—1,275, disease death/total 2,538/3,870 (65.6%); Disease-related deaths, 1618-24—2,538; Other causes of death or return to England—1,332; Total—5,145.

\* Includes deaths from all causes as well as those returning to England alive.

† Since 347 known deaths occurred in the massacre, the remainder are assigned to disease.

‡ Overall death rate fell below the disease rate, hence all were assigned to disease.



population grew from 1,210 to 4,914, while receiving an estimated 9,000 immigrants.<sup>[1]</sup> Although over half of the population died in the 9-year period, this figure obscures the marked improvement in annual mortality. Had the pre-1624 mortality rate of 28.3 per cent per year continued, Virginia in 1634 would have numbered 2,456 instead of 4,914. In effect, annual mortality was cut in half (to about 14.2 per cent) during the early royal period (Table 5).<sup>[2]</sup>

TABLE 5  
*Population growth 1625-34 under pre-1625 disease mortality rates and a fitted mortality rate*

Date	Pre-1625 disease Mortality rate (28.3% per year) Population and immigrants	Survivors	Fitted mortality rate (14.2%) for 1625-34 Population and immigrants	Survivors
1625-6	1,210 + 1,000	1,582	1,210 + 1,000	1,896
1626-7	1,582 + 1,000	1,849	1,896 + 1,000	2,485
1627-8	1,849 + 1,000	2,040	2,485 + 1,000	2,990
1628-9	2,040 + 1,000	2,177	2,990 + 1,000	3,423
1629-30	2,177 + 1,000	2,275	3,423 + 1,000	3,794
1630-1	2,275 + 1,000	2,344	3,794 + 1,000	4,113
1631-2	2,344 + 1,000	2,394	4,113 + 1,000	4,387
1632-3	2,394 + 1,000	2,430	4,387 + 1,000	4,622
1633-4	2,430 + 1,000	2,456	4,622 + 1,000	4,824
1634-5	2,456 + 1,000	2,475	4,824 + 1,000	4,997

Actual population in 1634, 4,914. The above calculations assume no natural increase. Some children were born in the colony during the period, but the imbalanced sex ratio favouring males and other evidence suggest that children contributed little to population growth at this time.

Several factors caused the decline in mortality. By far the most important was the shift in population patterns. By 1634, the deadliest zone along the James, including James City, Warwick and Warrowerguyoake counties, contained 45 per cent of total population. But population had spread into healthier zones, including the lower York in the salt on the south side of the river, the fresh water at the head of the James and the lower James and the Eastern Shore.<sup>[3]</sup> The general dispersal of tobacco plantations within all ecological zones also favoured life. Typhoid and dysentery could not become epidemic when settlement was scattered.

[1] One thousand immigrants per year is the estimate of Morgan, *Slavery and freedom* 159. Morgan underestimates the magnitude of declining mortality rates after 1624. A lower death rate is not inconsistent with his literary evidence. Ship captains experienced high mortality (42 per cent in 1636) precisely because they plied in the oligohaline zone. And 1,800 deaths in 1636, given the population and increased immigration in that year, produces a mortality rate in between that of the period 1618-24 (28.3 per cent) and our post-1624 estimated rate (14.2 per cent)

[2] Morgan's 1625-40 estimate of 1,000 immigrants per year has been questioned as too high by Menard. He suggests that immigration varied directly with tobacco prices, and therefore Virginia immigration peaked at about 2,000 in 1635-6 and in the preceding years (1625-35) immigration generally fell substantially below 1,000 per year. If Menard is correct, than annual mortality for the period 1625-34 falls even lower than the 14.2 per cent presented here. Russell R. Menard, *economy and society in early colonial Maryland* (unpubl. Ph.D. dissertation, Univ. of Iowa 1975) 167-70

[3] A list of the number of men, women and children . . . within the colony of Virginia, Anno Dne, 1634 *Colonial Records of Virginia* 91

And as population grew, settlement progressed inland away from the deadliest parts of the James and onto the hills, ridges and drainage divides where fresh springs provided a safe water supply. Dietary habits probably changed too, with increased consumption of wine, beer and cider instead of water, and reduced consumption of oysters and clams during May to August when these bivalves concentrated micro-organisms. As the "new healthiness" of the country took hold, Virginians and their visitors usually attributed it to climate, improved by forest clearance. More accurately, the spread and dispersal of settlement along with certain dietary adjustments were the keys to life.<sup>[1]</sup>

Frontier expansion and plantation dispersal continued during the rest of the seventeenth century, with generally beneficial demographic effects. Although travellers commented on the pallid complexions, sickness and death in the salt-water environments of the lower estuaries, the population there was generally much healthier than in the fresh-salt transition.<sup>[2]</sup> As the frontier expanded into

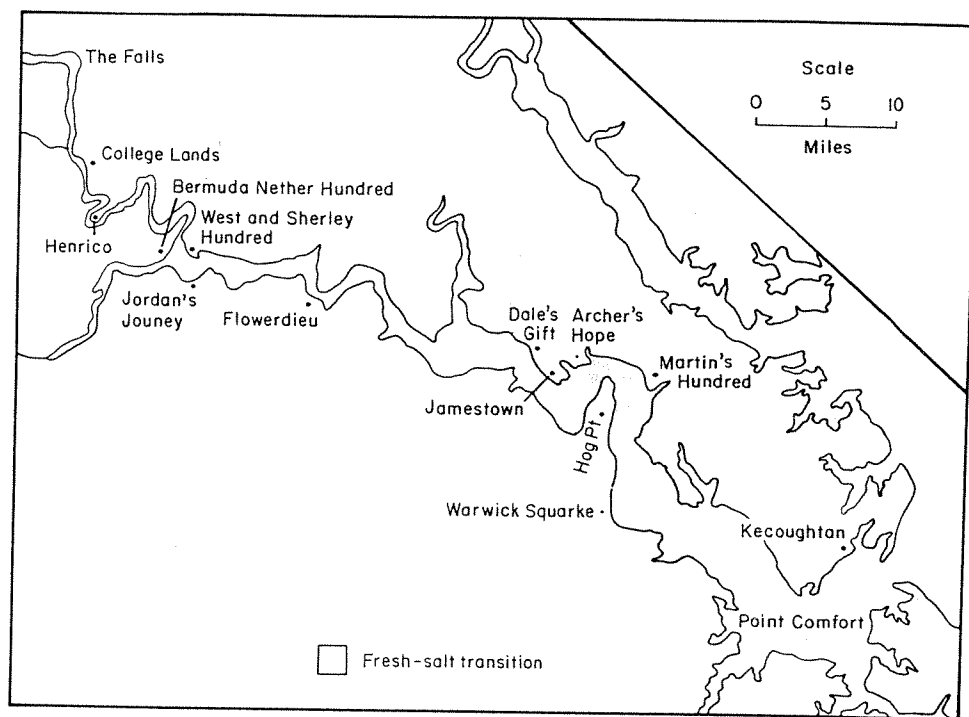


Figure 1. The James River, 1607-24.

- [1] On dispersal and tobacco, see Morton, *Colonial Virginia I*, 122-33; Hecht, "The Virginia colony" 195-207; Craven, *Dissolution of the Virginia Company* 170-1. In a revealing note, Governor Wyatt in 1623 blamed the colony's ill-fortune on "the intemperate drinking of water". "To plant a colony by water drinkers was an inexcusable error in those who laid the first foundation and have made it a received custome". *Kingsbury Records IV*, 10-1, 453; Wharton, *The bounty of the Chesapeake* 46; William Ancisz and C. B. Kelly, Self-purification of the soft clam *Mya arenaria* *Public Health Reports* 70 (1955) 605-14
- [2] Gilbert Chinard (Ed.), *A Huguenot exile in Virginia, or voyages of a Frenchman exiled for his religion with a description of Virginia & Maryland* (New York: The Press of the Pioneers, Inc. 1934) 130, 174. Recognition of the fresh-salt transition and its dangers is incipient in Bullock's "flowing of the salt". Clayton thought all salt water bad, as it impregnated the air and thus damaged the human body. William Bullock, *Virginia impartially examined . . .* (London 1649) 4; Edmund and Dorothy Smith Berkeley (Eds.), *The Reverend John Clayton . . . his scientific writings and other related papers* (Charlottesville: University Press of Virginia 1965) 54

the oligohaline zones of the York, Rappahannock and Potomac rivers, death rates rose above those in either the salt or the fresh-water zones. The spatial pattern of mortality, as reconstructed from a Virginia parish census of births and deaths for 1725-6, tends to confirm the persistence of disproportionate death in the oligohaline (Figs 2 and 3).<sup>[1]</sup> Along the four principal Virginia estuaries, the pattern of mortality hypothesized by the estuarine model for early Virginia remains recognizable a century later. Progressing down river, the proportion of burials to births is generally least in the fresh water, peaks in the oligohaline and drops slightly in the salt water of the lower estuaries. Left bank (north side) mortality is usually higher than the corresponding right bank (south side). The map's subtleties and its several departures from the model—notably the higher than expected mortality in the upper Rappahannock as well as in Nansemond County in the salt water of the lower James and the rather higher levels of mortality along the Potomac estuary as compared to the other valleys—warrant more attention, but to do so would push us beyond the limited scope set out for this essay. Although the chances for survival in the Chesapeake had improved dramatically after 1624, the mortality map of a century later strongly implies that typhoid and dysentery continued as an important cause of death, particularly in the zone of the fresh-salt transition and, to a lesser extent, in the zone of salt water.<sup>[2]</sup>

The demographic history of early Virginia is both sad and tragic. Sad because so many died; tragic because they died needlessly. Smith, Dale and others knew

[1] The census records births and burials for the year beginning 15 April 1725. I have assumed that census recording procedures were uniform among Virginia parishes, but undoubtedly these procedures varied markedly. More thorough studies of Virginia demography may reveal whether the parishes departing from the estuarine-disease model reflect actual differences or recording biases in the census. Public Record Office, C.O. 5/1320, f. 74. Parish boundaries are roughly accurate. The four Henrico County parishes are aggregated on the map. Charles Francis Coke, *Parish lines diocese of Virginia*, Virginia State Library Publications 28 (Richmond: Virginia State Library 1967); George Carrington Mason, *Colonial churches of tidewater Virginia* (Richmond: Whittet and Shepperson 1945); the fresh-salt transition zones are located according to Nichols, "Sediments of the James River" 171-9. H. C. Whaley and T. C. Hopkins, *Graphical summary reports Nos. 1-2: Atlas of the salinity and temperature distribution of Chesapeake Bay*, Chesapeake Bay Institute, The Johns Hopkins University, Reference 52-4 and 63-1 (Baltimore: 1952 and 1963); Chinard, *A Huguenot exile* 174

[2] This geographical pattern of mortality might be explained by other models, such as the Rutman's malarial endemicity. According to the Rutman's, malarial "morbidity climbs as endemicity rises, since a greater percentage of infectious bites by *Anopheles* leads to symptomatic malarial attacks. Yet the rate of morbidity will be balanced at some point by the rate of immunities in the population and then will begin to decline until, in a hyperendemic situation, morbidity is largely limited to children, non-immune newcomers to the community, and pregnant women". Put geographically, this process of endemicity should move roughly with the frontier of settlement, i.e. old settled areas being hyperendemic, newly settled areas having low but rising morbidity, and middle-aged areas having very high morbidity. If I have reasoned correctly, the entire James River area, as the oldest settled zone, should show similarly low values on our map, followed by very high values in the middle-aged tier of Gloucester, Middlesex, Lancaster, Westmoreland and Northumberland counties, and low values elsewhere. I do not detect such a pattern, and accordingly favour the three-zone estuarine model of enteric diseases. Rutman and Rutman, "Agues and fevers" 37-9, 44-5. Also, Lorena S. Walsh and Russell R. Menard, "Death in the Chesapeake: two life tables for men in early colonial Maryland" *Maryland Historical Magazine* LXIX (1974) 211-27; and Robert V. Wells, *The population of the British colonies in America before 1776* (Princeton: Princeton University Press 1975)

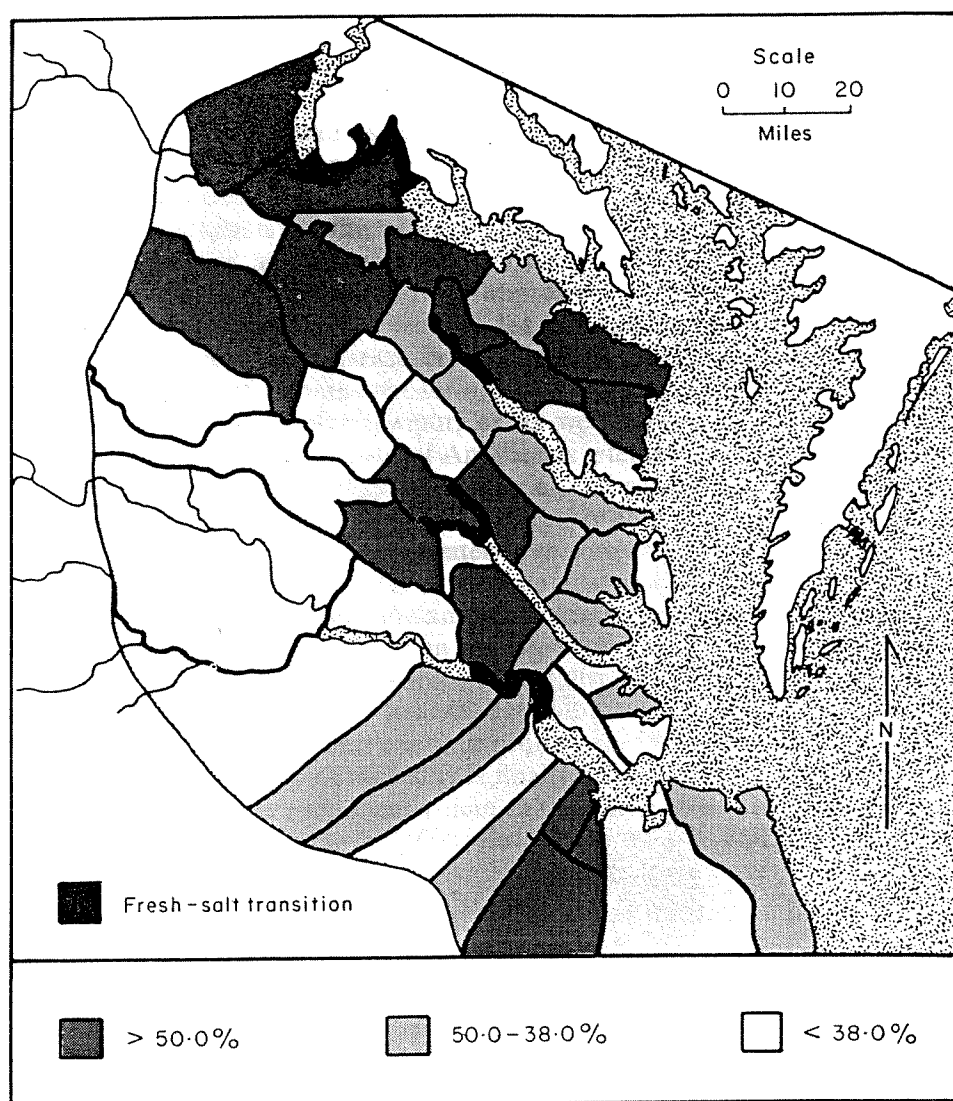


Figure 2. Proportion of burials to births, 1725-6: Virginia parishes.

that epidemics of typhoid, dysentery and salt poisoning were recurrent; they knew that these epidemics were spawned by a contaminated water supply in the vicinity of Jamestown and the fresh-salt transition on the James estuary. They knew that dispersing in the summer or shifting settlement permanently into the fresh-water zone were the only ways to save lives. And they knew that scattered settlements required the protection of an aggressive Indian policy. Smith and Dale saved lives, but their insights were abandoned with the arrival of new colonial leaders or a new Company administration. Jamestown was reclaimed, mortality rose, and the painful environmental learning process began at ground level. The Sandys administration never learned. The nexus of environment and mortality confounded and eluded them. They mistakenly believed that the "seasoning" process would eventually take hold and Virginia's population would grow. But typhoid and dysentery were no respecters of flawed theories of immunity. From a demographic



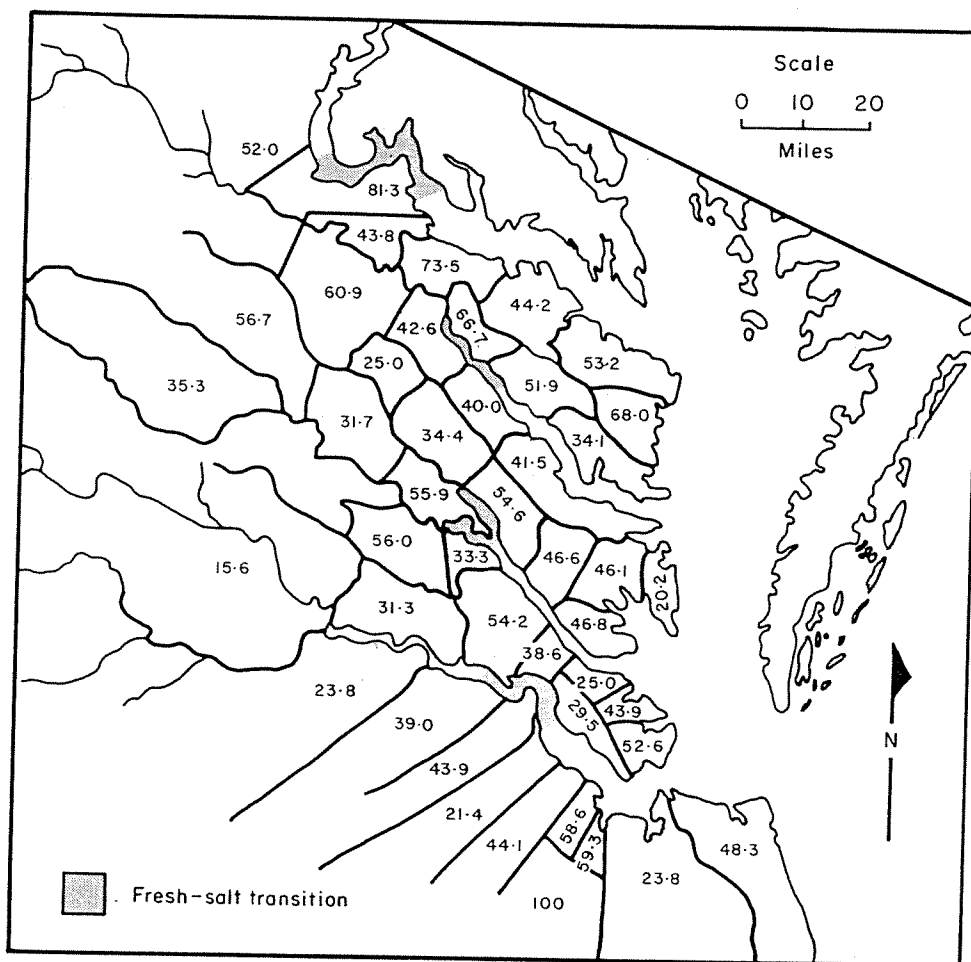


Figure 3. Proportion of burials to births, 1725-6: Virginia parishes.

standpoint, the best thing that happened in early Virginia was the dissolution of the Company with its fixation on Jamestown.

Never again would Englishmen in America occupy such a persistently deadly location as early Jamestown. Indeed, across the entire sweep of British expansion, only Calcutta and the West African slave ports match the levels of mortality in early Virginia.<sup>[1]</sup> North American colonization was, in fact, remarkably healthy after 1624. Epidemics of enteric disease such as those in early Virginia were virtually unknown during the rest of the seventeenth century. In large measure, this was so because the English took greater care in the selection of town sites. Salty ocean or bay water virtually surrounded the towns of Boston and Newport in New England and Charlestown in Carolina. These briny locations minimized disease epidemics because the salt water was, in the first place, undrinkable and

[1] Philip D. Curtin, *Epidemiology and the Slave Trade* *Political Science Quarterly* LXXXIII (1968) 190-216; and Rhoads Murphey *The city in the swamp: aspects of the early growth of Calcutta* *Geographical Journal* CXXX (1964) 241-56; and W. B. Morgan, *The influence of European contacts on the landscape of Southern Nigeria*, pp. 193-208 of R. Mansell Prothero (Ed.) *People and Land in Africa South of the Sahara* (New York: Oxford University Press 1972)

because river and tidal action flushed out the pathogens and lowered their concentrations in the surrounding bays and ocean. Moreover, at each of these town sites, drinking water was supplied by excellent and steady springs, located usually at higher elevations. Equally salubrious was the site of Philadelphia, located in the "freshest" of the Delaware River beyond the reaches of the deadly oligohaline.<sup>[1]</sup>

These healthier locations were not felicitous accidents; rather they reflected a heightened perception of the more extreme New World environment and of estuarine dynamics and water supply. The settlers of Massachusetts in 1630 exemplify a striking change in their approach to the environment. Whereas Virginians struggled through 17 years of death at Jamestown, the Puritans learned more rapidly. Soon after disease broke out in Charlestown (Massachusetts) in 1630, the majority of Puritans left for other sites, particularly the salt enshrouded and elevated peninsula of Shawmut, or as we know it today, Boston.<sup>[2]</sup> The Puritans abandoned the analogue of America as a temperate marine environment; instead, their locational strategy relied on trial, error and rapid negative feedback. Geographical location, guided by practice rather than theory, afforded the best preventive medicine against enteric water-borne diseases in the dynamic American coastal environment. Henceforth, densely populated English settlements avoided the zone of the fresh-salt transition and thus escaped the epidemics of typhoid and dysentery that ravaged Jamestown in the late summer.

We have not heard the last word on mortality in the oligohaline. These predominantly rural zones will, I suspect, achieve a cruel prominence as historical demographers and geographers continue probing the geography of early American mortality. When the map of mortality is finally drafted, the oligohaline will stand out and nowhere more prominently than in the marshes and tidal swamps in the wet rice districts of Carolina and Georgia. Where the ebb and flow of fresh and salt water were integral parts of wet rice culture and where slaves<sup>[3]</sup> were unable to escape from the lethal environment, the spectre of disease and death is sadly predictable.<sup>[4]</sup>

*Department of Geography,  
University of Maryland  
Baltimore County*

[1] Earle "The first English towns" 34-50; Carl Bridenbaugh, *Cities in the wilderness: urban life in America, 1625-1742* (New York: Capricorn Books 1964); Darrett B. Rutman, *Winthrop's Boston: a portrait of a Puritan town, 1630-1649* (Chapel Hill: University of North Carolina Press 1965)

[2] Rutman, *Winthrop's Boston* 23-40

[3] Duffy, *Epidemics in colonial America*. The recent suggestion that slaves in South Carolina were relatively more immune to yellow fever and malaria than were white Europeans may have given blacks an advantage in wet rice cultivation; however, slaves would not have been immune to water-borne typhoid or dysentery which probably were very common in the wet rice fields where they worked. Peter H. Wood, *Black majority: negroes in colonial South Carolina from 1670 through the Stono rebellion* (New York: Alfred A. Knopf 1974) 63-91; on the use of fresh and salt water flows in the tidal rice fields, see Sam B. Hilliard, *Antebellum Tidewater Rice Culture in South Carolina and Georgia* pp. 91-115 of James R. Gibson (Ed.), *European Settlement and Development in North America: Essays on Geographical Change in Honour and Memory of Andrew Hill Clark* (Toronto and Buffalo: University of Toronto Press 1978)

[4] In preparing the final version of this paper, I was aided immeasurably by the comments of David Ammerman, Daniel Doeppers, James Knox, Allan Kulikoff and Russell Menard.